To: Mr. Edward Hanlon, Designated Federal Officer (DFO) for the Radiation Advisory Committee, EPA Science Advisory Board Staff Office (1400R), U.S. Environmental Protection Agency, submitted via email to hanlon.edward@epa.gov.

Beyond Nuclear thanks EPA's Office of Radiation and Indoor Air (ORIA) for the opportunity to provide written comments to the Science Advisory Board Radiation Advisory Committee (RAC) regarding EPA's Advance Notice of Proposed Rulemaking (ANPR) to consider revising the Environmental Radiation Protection Standards for Nuclear Power Operations (40 CFR part 190), as allowed under the Federal Advisory Committee Act (FACA).

November 3, 2015

If EPA ORIA does decide to update its 1977 radiation exposure standards, there are at least five key issues on which the RAC should advise EPA so that EPA may be better able to protect the public, *particularly pregnancy and early childhood life stages*, from exposure to radioactivity.

1. **The RAC should help EPA use existing frameworks to establish regulations that *fully* protect the most vulnerable life stages, despite remaining uncertainties of radiation's impact on early human development.**
2. **Since the RAC's charge is to advise ORIA on this potential rewrite of radiation standards, RAC should make a special effort to emphasize protection against unique impacts to early life stages. To this end, RAC should consult with, or offer committee memberships to, pregnancy and/or child health and development experts who are familiar with impacts of toxic chemicals or radioactive isotopes on these vulnerable stages. Greater representation of scientific disciplines such as genetics, evolutionary biology, and ecology might also provide a benefit to this committee as it aids EPA ORIA in its charge.**
3. **If EPA decides to integrate recent ICRP recommendations into new exposure standards, the RAC should aid EPA in overcoming the deficits in ICRP assumptions. Not only does ICRP fail to specifically account for some unique vulnerabilities that occur during developmental life stages that could result in non-cancer diseases, but studies of childhood cancer risks indicate that current ICRP exposure limits for the in utero life stage are not protective enough overall.**

4. **RAC should help guide EPA to protect for genetic impacts of radiation exposure past the second generation since the ICRP model doesn't extend that far. EPA should not assume that, although cancer may carry the greatest impact most immediately, genetic and epigenetic impacts won't surpass this risk in a few generations in the wake of incrementally increasing chronic, low dose exposure.**
5. **RAC should point to concrete ways EPA can combat the "magical thinking" in radiation health assessment that believes dose estimation is much more reliable than it actually is. This belief remains steadfast—even if radiation-associated diseases increase in a population, unreliable dose estimates are used to claim radiation bears no responsibility. These dose estimations have often been based on averaged radiation releases measured by industry. Measurement of these releases has been fraught with secrecy and improper measurement technique, resulting in unrealistic exposure scenarios that are then heavily relied upon for health impact assessments to the detriment of public health.**

1) The RAC should be aware of frameworks that guide EPA regulations with regard to early life stage exposure to toxins and carcinogens. ORIA's reconsideration of the 1977 radiation exposure standards should follow these established frameworks.

A) President Clinton's Executive Order 13045 of April 21, 1997 is still in force. The *Protection of Children From Environmental Health Risks and Safety Risks* states "...each Federal agency: (a) shall make it a high priority to identify and assess environmental health risks and safety risks that may disproportionately affect children; and (b) shall ensure that its policies, programs, activities, and standards address disproportionate risks to children that result from environmental health risks or safety risks." Any regulatory changes should protect for these life stages.

B) EPA's 2005 Children's Health Guidance divides birth through age 20 into life stages.[1] Other life stages may be important "when assessing human exposure and risk including: pregnancy; nursing, and old age." The 2005 Children's Health Guidance states

"consideration is given for risks resulting from fetal exposure via direct exposure to the pregnant mother, as well as risks due to postnatal exposures."[2]

C) EPA Supplemental Guidance for Early-Life Exposure to Carcinogens recommends a 10-fold susceptibility factor be applied for risks to children up to 2 years of age.[3] In the case of childhood leukemia the onset is likely to be in utero, and the 10-fold factor may be orders of magnitude greater for the embryo.[4]

2) Little research exists on realistic, chronic, very low dose exposure impacts on pre-implantation, the developing human embryo and developing fetus. These early life stages and growing children have special vulnerabilities, including predilection for certain types of radionuclides, particularly ones that mimic chemicals used to form constituents of growth and aid metabolism. Models that only represent radiation damage to organs, the whole body, or even groups of cells, may not fully represent the extent of damage to the individual cells that are required for healthy in utero development. The RAC should help EPA to work around any uncertainty about the impact of radioactivity on pregnancy and childhood, with the ultimate goal being full protection of humans. No one gets to adulthood without first being a child, so continuing to expose humans during these vulnerable life stages could have far-reaching consequences for individuals and populations.

Intrauterine programming, a recently discovered phenomenon that is just now being researched, offers insight into the vulnerability of the in utero life stages that can impact the wellness and viability of not only those life stages, but also subsequent adult wellness and viability as well.* Much about this phenomenon is not yet discovered, but this uncertainty should not be used as an excuse to expose humans to undetermined damage from radiation during the most vulnerable stages of life.

Pregnancy begins before conception;[5] therefore women of childbearing years should be treated as potentially pregnant before being aware of a pregnancy. And since a female fetus develops all the eggs she will ever have while she is in utero, protecting pregnancy from

* DNA damage and epigenetic changes in the embryo can manifest permanent structural and functional changes and can lead to other diseases later in child or adult life stages, i.e. diabetes, cardiovascular disease, and premature aging. http://embor.embopress.org/content/embor/11/1/32.full.pdf

radiation is protecting not just the child of the pregnant woman, but her grandchildren as well. It is worth noting here that *adult women* are 50% more likely than men to get cancer from the same amounts of radiation, according to the information in the BEIR VII report,[6] although these comments focus almost exclusively on *early* human life stages.

The placenta is a temporary but immensely important structure that performs organ-like functions during pregnancy. It supplies oxygen and nutrition to the embryo/fetus and removes metabolic products as well as providing a limited barrier against some toxins and drugs; it is active endocrinologically to support the ongoing pregnancy.[7] "...Radiation affect[s] fetal growth not only by damaging fetal cells but also by impairing placental development and function by the induction of apoptosis and cell cycle arrest in trophoblasts... In human pregnancy, apoptosis is increased in placentas subjected to intrauterine growth retardation or other disorders."[8]

The in utero life stage and childhood can have disproportionate vulnerabilities to certain radionuclides.[9] Among these are radioactive carbon (C-14) and tritium (radioactive hydrogen isotope). † Each of these can collect in fetal tissue to twice the concentration in maternal tissue. This concentration factor serves as an additional risk to the sensitivity of this life stage. Further, there is indication that the type of radiation given off by these isotopes—a beta particle—could have more impact than is currently assumed.[10,11,12,13] Children and adolescents exposed to radioactive iodine (iodine-131), and who did not receive a protective measure of potassium iodine within a few hours of exposure, not only got cancer, but were found to have more aggressive forms of cancer.[14]

Radionuclides *inside* a developing embryo, or the developing placenta surrounding it, could do unique and tremendous damage. "[T]he hazard from internal radiation exposures increases markedly with younger people."[15] Cancer, by either promotion or induction, is only one disease endpoint. While young children are more sensitive to radiation compared to adults—ICRP recognizes that they can be more sensitive—the beginning stages of life (embryonic and fetal), which are unique in structure and development, can at some stages be many times more sensitive than even young children, due to phenomena like intra-uterine programming and organ creation.

† Sr-90 also collects more during periods of growth: Frequently asked questions: Strontium 90. Delaware Health Services. January 2012

3) EPA ORIA asks in its ANPR "In updating the dose standard, should the methodology in ICRP 60 or ICRP 103 be adopted…" Accepting the methodology in ICRP 60 or 103 would essentially leave out protection of pregnancy because ICRP fails to specifically account for some unique vulnerability that occurs during this developmental life stage. Further, studies of childhood cancer risks indicate that none of the ICRP exposure limits for the in utero life stage are protective enough. A number of studies indicate childhood leukemia risks are elevated in studies of young children living near normally operating nuclear facilities or in environments of background radiation considered within the range of normal. RAC should aid EPA in overcoming the deficits in ICRP assumptions and recommendations if EPA decides to update the existing standards using ICRP reports.

Unfortunately, in several specific ways ICRP's large body of multi-decadal radiation exposure recommendations fails to offer adequate protection for pregnancy or pre-natal life stages.

ICRP assumes "that life-time cancer risk following in utero exposure will be similar to that following irradiation in early childhood,"[16] and relies on doses to the maternal uterus to assess dose to the embryo (which ICRP defines as up to 8 weeks from conception)[17]. However, "haematopoietic tissues appear more radiosensitive in embryos/fetuses than in newborn babies."[18] The heart, spinal cord and brain, major blood vessels and the beginning development of bones and muscles, are in process of forming from single cells, meaning that assessing damage from radiation during this sensitive stage presents unique challenges under circumstances not yet fully understood. This life stage deserves protection none-the-less.

ICRP does not assess damage to the placenta, a temporary but, immensely important structure that performs organ-like functions during pregnancy.[19,20]

ICRP states that for radionuclides ingested by the mother, "…doses to the embryo, fetus, and newborn child are similar to or less than those to the Reference Female."[21] The *exposure* may be similar, but ultimately, even what is considered a small dose could be much more

damaging for in utero development and could increase susceptibility to disease in adulthood because of intrauterine programming. ICRP is partially responsible for a latency time of 40 years to integrate new knowledge of radiation damage into protection policy. This could be, in part, because ICRP has been historically unwilling to acknowledge, let alone accommodate, newly discovered forms of damage without a *proven* mechanism, even though the science points to a *plausible* connection between this damage and radiation exposure. Regulatory bodies often adopt ICRP recommendations (which already lag behind the latest science) often a decade after they are released. Therefore, the latest phenomena that indicate heretofore-unrecognized forms of radiation damage -- bystander effect, genomic instability, and now intrauterine programming--may take 4 decades to achieve ICRP recognition and be integrated into exposure regulations.[22] EPA has to protect the public to a better standard by using the newest data in addition to, or instead of, relying on older studies and recommendations.

ICRP makes no attempt to specifically protect *female* fetuses, which develop all the eggs that child will ever have during prenatal growth. Therefore, radiation exposure during the development of a female fetus could impact *her* children, making this female embryo or fetus the most vulnerable life stage and her damage potentially cross-generational. This means that exposing a female of childbearing age to radiation could impact her grandchildren, depending on the timing of her pregnancy. Many women do not know they are pregnant, but crucial pregnancy development is still occurring. Likewise, doctors can usually not tell the sex of the fetus until 16-20 weeks after conception—and after the fetus is well into developing its own sex organs. Therefore, every fetus, even though the sex is biologically defined at conception, needs to be protected as a female because its sex cannot be determined by examination until much later in its development.

And because ICRP ignores critical sensitivities during pregnancy, and also fails to account for the most recent research on realistic risks of radiation exposure during early human life stages, doses ICRP claim are protective of pregnancy pose unrecognized risk. It is clear that our early human life stages bear a disproportionate burden from exposure to radioactivity.

Many radiation studies examine impacts on laboratory animals or special cell lines outside of the body. While such studies can indicate what *kind* of damage from

radiation could be expected, a direct comparison between these studies and realistic exposure scenarios from routine or catastrophic releases of radioactivity is tenuous at best for a number of reasons: 1) doses used in the laboratory are often much higher than dose exposures expected from routine releases or from eating food contaminated with small amounts of radiation; 2) laboratory doses are rarely given over a large span of time, which would be needed for assessing whole life or cross-generational damage; and 3) recent research that compared impacts of chronic radiation exposure around Chernobyl with radiation experiments done in controlled environments, indicates that wildlife in nature could be *at least* eight times more sensitive to radioactivity than animals used in controlled experiments.[23] For these reasons and others, studies relied upon for assessment of radiation damage must be carefully chosen, especially when used for health assessments investigating vulnerable human life cycles.

Research summarized below addresses a number of the concerns stated above in that they are *in vivo*, non-laboratory conditions. Many examine real-life, protracted exposure to actual low-doses of radioactivity.

Childhood cancer from natural and man-made background radiation

Two studies, one examining impact of natural background radiation (Kendall, et al.) and one examining both natural background radiation‡ and background radioactive cesium from Chernobyl (Spycher, et al.), indicate a statistically significant increase in childhood leukemia from approximately a 4 mSv (400 mrem) *cumulative* dose (see Figures below). At 1.3 mSv per year, an increased hazard begins to appear for all childhood cancers and continues to increase as the dose increases, reaching just about double at 2 mSv per year. Central nervous system (CNS) cancers and leukemias seem particularly sensitive. (see Figure below from Spycher, et al.). These studies have high statistical power.[24] The amount of radiation, and the risks it poses to early life stages, is well within even natural background levels received in 1 to 4 years (80-100 mrem per year, not including radon—see footnote). [25,26] Leukemia induction at these levels (1 mGy per year) is supported by other studies as well.[27] Any exposure to man-made radioactivity will increase an already naturally elevated cancer risk to childhood life stages.

‡ Radon dose is excluded from these studies because it does not deliver its dose to the bone marrow, thought to be the seat of leukemogenesis. Radon is also mitigable.

Figure S2 from Spycher, et al. 2015

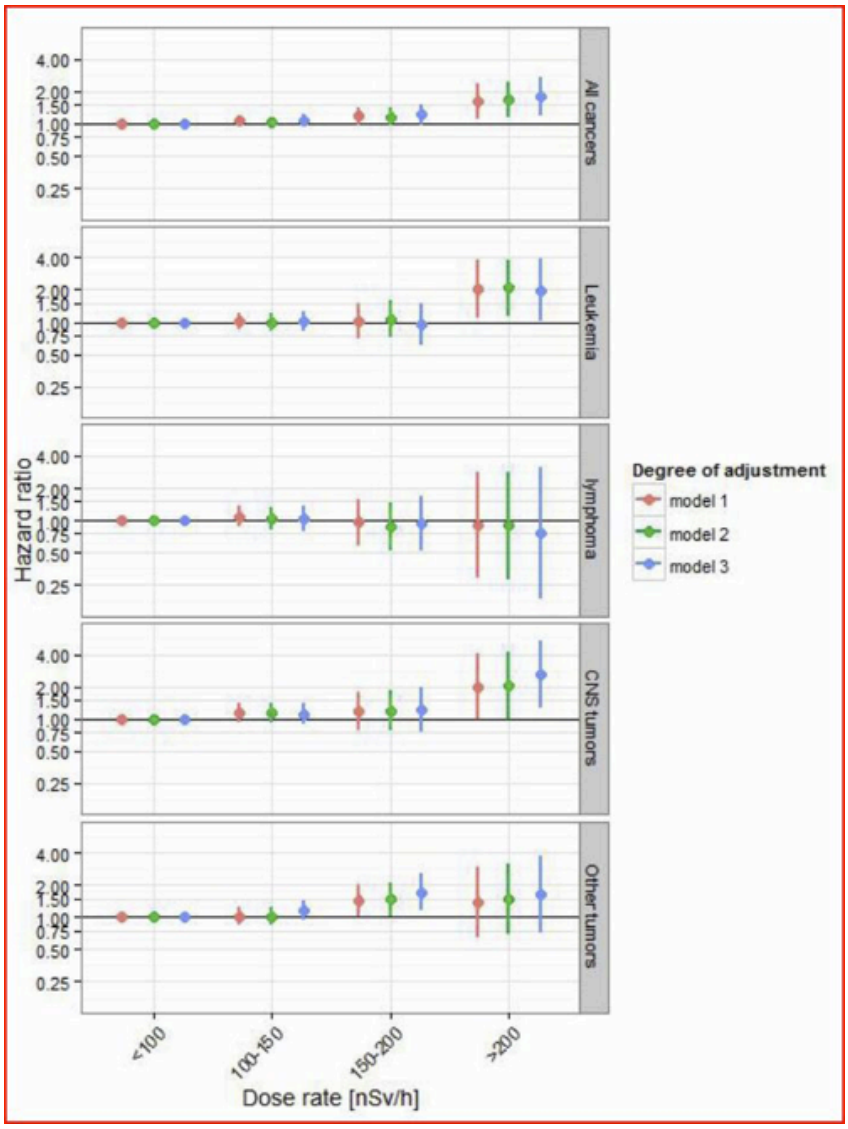
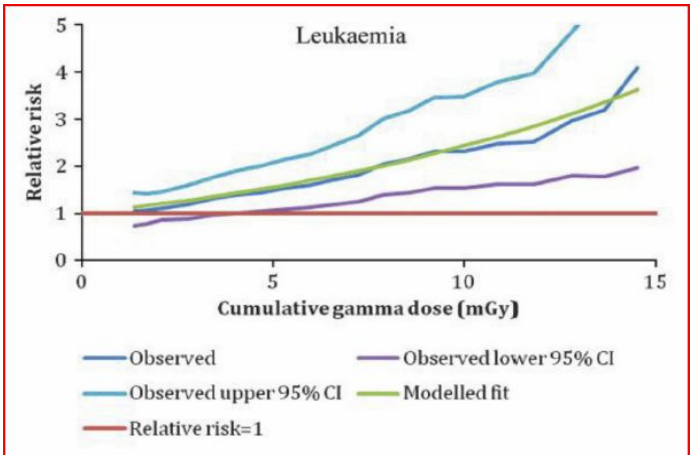
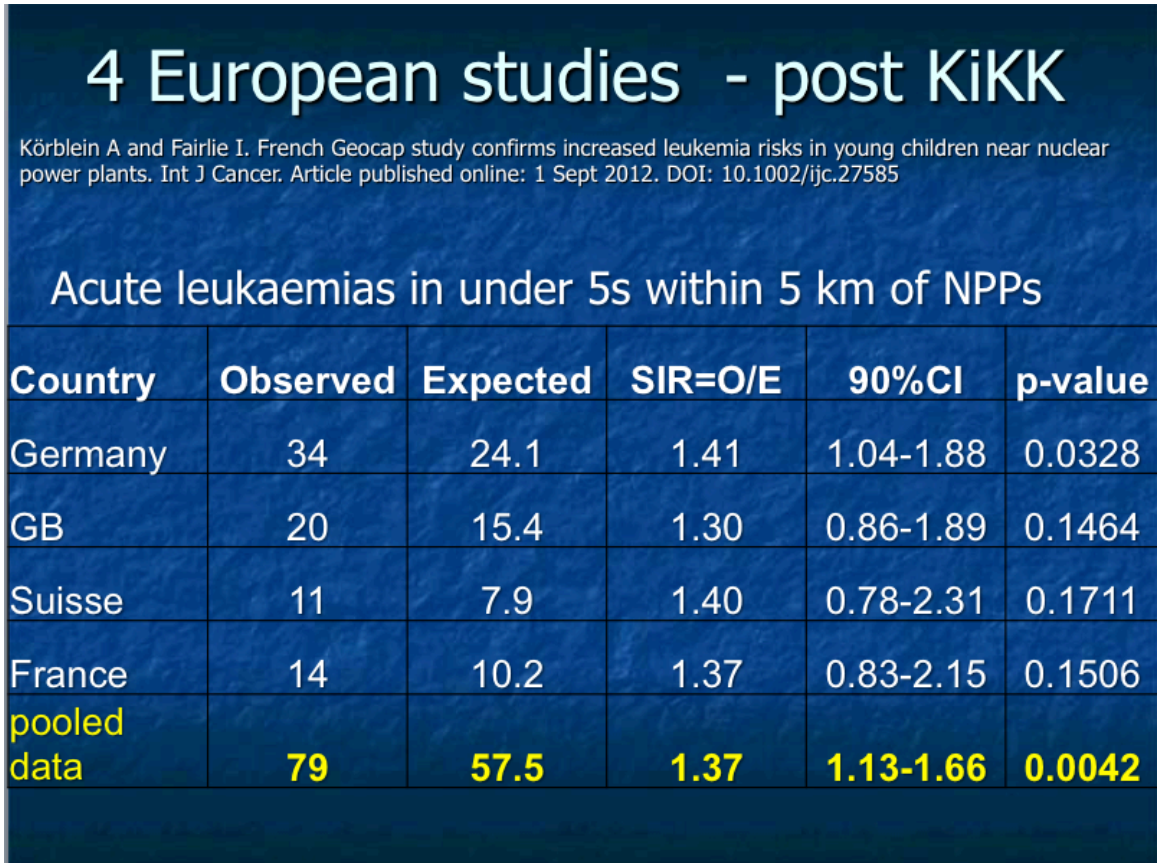


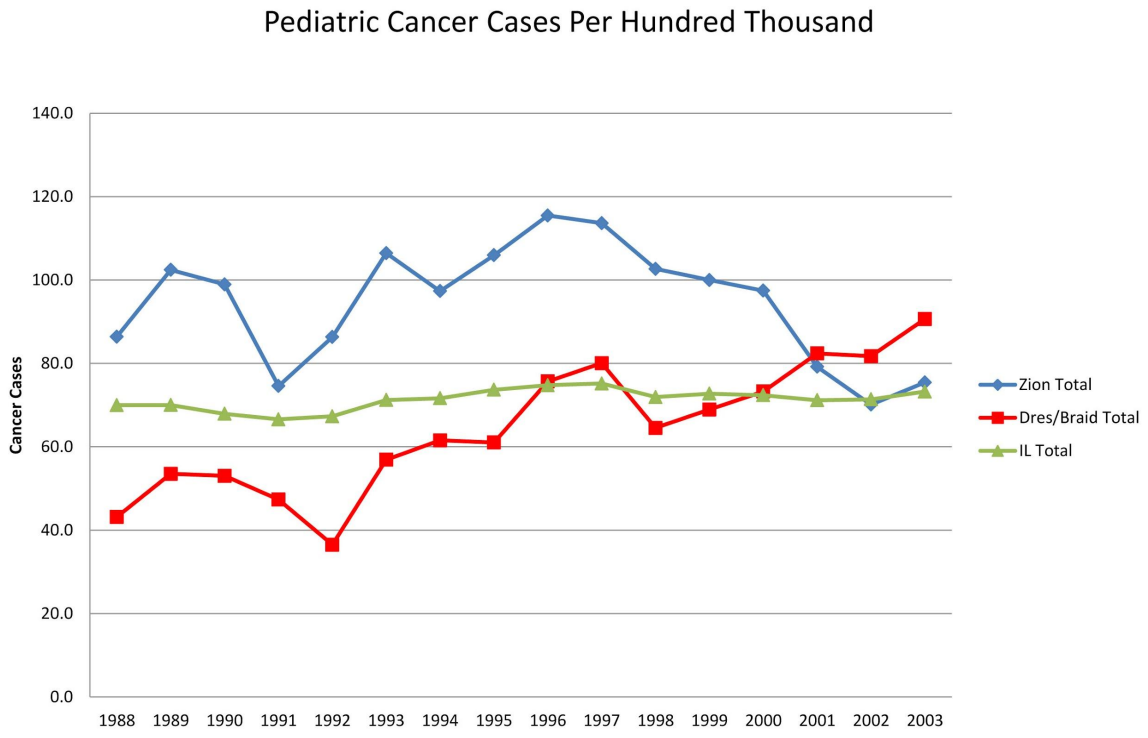
Figure 1 from, Kendall, et al. 2013



Increased childhood leukemia around nuclear reactors worldwide

"Over 60 epidemiological studies world-wide have examined cancer incidences in children near nuclear power plants (NPPs): most of them indicate leukemia increases." This would be an unexpected health impact, because the doses these children received, as estimated by nuclear power operators, were well below those expected to result in disease—by a factor of 10,000.[28] Although the reason for this very large discrepancy is unknown, uncertainties about the risks posed by certain radionuclides, as well as unaccounted-for sensitivities of certain life stages, could offer explanation. Because of these uncertainties, the existence of this effect in a large number of studies must be treated as real, and radiation dose reconstruction must be treated with skepticism, particularly for in utero effects. This effect is most pronounced in children under 5 years of age living within 5 km of a normally operating nuclear facility (see chart below compiling study results in the under 5s within 5). If study data of this age group is absorbed into older children, or a wider geographic area, this effect of radiation will be watered down or hidden altogether.[29]





Graph from Dr. Joseph Sauer's presentation at NAS Hearing in 2011[28]

Pediatric cancers around nuclear facilities in Illinois, USA

Dr. Joseph Sauer, a practicing physician, living in Illinois near the Dresden and Braidwood nuclear power stations, obtained Illinois Department of Health statistics segmented by county and zip code for pediatric cancers in Illinois from 1988 to 2003. In the figure below, Dr. Sauer presented his findings at the National Academy of Sciences, Analysis of Cancer Risks in Populations Near Nuclear Facilities, Phase 1 hearing in Chicago in 2011.[30] Dr. Sauer's slide from his presentation[31] is shown below.

The curve represents total pediatric cancer cases per population total of 100,000 in each year in Illinois; the cases in counties surrounding the Zion station (shut down in 1998); and the cases in counties surrounding the Dresden (started operation 1970 and continues to present) and Braidwood (started operation 1988 and continues to present) stations. This data suggests an approximate excess of 40 cases per population of 100,000 per year around the time Zion was shut down. This health outcome data was analyzed in the context of particular citizen concern regarding unplanned tritium releases from Braidwood and is higher than the EPA least protective cancer risk goal of 1 in 10,000.

Significant negative non-cancer effects from natural background radiation

A meta-analysis examining impacts of living in areas where the natural background levels of radiation are high, found extensive evidence of increases in "significant negative effects on immunology, mutation and disease frequency", including reduced levels of antioxidants. The effects were small, but consistent and significant. "Note, however, that there is no evidence of radio-tolerance or radioresistance in humans…" Mutation effects, including but not limited to cancer, start at approximately 1 mSv per year of natural background radiation.[32] Another meta-analysis "suggests a strong impact of radioactive contamination on individual fitness in current and future generations, with potentially significant population-level consequences, even beyond the area contaminated with radioactive material." Plants, humans, and non-human animals were included in this study.[33]

Organ damage from radiocesium inhaled or ingested

In studies of post-Chernobyl Belarus, cardiac abnormalities develop in children whose bodies contain 10-30 Bq/kg of radioactive cesium. In addition to heart arrhythmias, the radioactive cesium disrupted the energy of cardiac cells in turn decreasing the child's ability to adapt to, and pull through, everyday stresses including physical and mental pressure, infections, allergies and more. Tissue death and irreversible pathologies develop at 50 Bq/kg.[34,35]

Obstructed lung capacity associated with radioactive cesium contamination from Chernobyl

Two studies demonstrate that obstructive and restrictive lung function was associated with radioactive cesium 137 contamination. The first study[36] examined a pediatric cohort residing in the Narodichesky district of Ukraine from 1993 to 1998 and found an association with radioactive cesium in the soil. Researchers conducted a follow-up study to see if this effect persisted by examining children 8-17 years old, born in and living within differentially contaminated areas around Chernobyl. The mean internal dose estimate of 0.165 mSv/yr was derived using whole body counters. This follow-up study concludes that "Children in a region just outside of the closed Chernobyl contamination zone continued to

have respiratory health deficits associated with ^{137}Cs whole-body burden as recently as 2010."[37]

Perinatal mortality after internal exposure to radioactive strontium from atomic testing and Chernobyl

Perinatal mortality increased in Germany, Belarus and Ukraine subsequent to the Chernobyl catastrophe, at the time radioactive strontium collected in the bodies of pregnant women. This effect persisted until the end of 1998, when the study ended. Additionally, "…about 80,000 excess early neonatal deaths in West Germany can be attributed to strontium from the fallout of atmospheric nuclear weapons tests."[38] Doses from atomic bomb fallout in Germany were well below 1 mSv/year, indicating that the ICRP assumption that the fetus is protected from certain kinds of damage by a radiation threshold dose of 100 mSv is wrong.[39,40,41]

Pre-implantation birth defects in areas contaminated by radiation from Chernobyl

A recent study demonstrates significantly increased rates of conjoined twins, teratomas, neural tube defects, microcephaly, and microphthalmia in an area of Ukraine polluted by ionizing radiation at levels higher than other places contaminated by Chernobyl. Many of these pregnancies end in termination with no guarantee that the reason for the termination (malformation) is properly listed, thus actual numbers of malformations could be underreported. The researchers characterize some of these effects as "blastopathies" which they define as "anomalies that arise prior to embryonal implantation and organogenesis…"[42]

Prenatal subclinical and clinical brain damage from Chernobyl radiation

Students born in regions of Sweden with higher Chernobyl fallout performed worse in secondary school, particularly in math. "Damage is accentuated within families (i.e., siblings comparison) and among children born to parents with low education… To the extent that parents responded to the cognitive endowment, we infer that parental investments reinforced the initial Chernobyl damage." This decreased performance in school was not accompanied by any health damage, meaning that neural development is compromised at

very low radiation doses (4 mSv is assumed to be the *highest* dose received by an individual in Sweden as a result of Chernobyl radioactivity). This very low radiation could be responsible for subclinical negative health impacts that would be harder to spot, much less attribute to a precipitating cause.[43]

Prenatal irradiation from Chernobyl has contributed to the deteriorated intelligence of children as shown by reduction of full scale and verbal IQ. This study suggests that prenatal exposure to ionizing radiation at fetal doses starting at 11 mSv can result in detectable brain damage.[44]

Childhood leukemia increases in areas contaminated by Chernobyl radioactivity

A case-control study examined acute leukemia cases in children who were in utero or under 6 years of age at the time of the Chernobyl accident. Estimated doses were less than10 mGy. "A significant increase in leukaemia risk with increasing radiation dose to the bone marrow was found. This association was most evident in Ukraine, apparent (but not statistically significant) in Belarus, and not found in Russia." The study authors conclude that this increase could be due to selection bias, but provide no evidence of this.[45] This assumption of selection bias was later shown to be unfounded through a detailed analysis of the original data.[46]

Exposure to chronic, low-dose radiation can increase radiosensitivity in successive generations

In addition to initial damage from radiation exposure, studies (summarized in 1998) of Chernobyl *animal* populations living in chronic low-dose radiation show an increase in radiosensitivity among those whose ancestors were exposed. This indicates that successive generations could be less able to cope with the same degree of exposure as their parents were and that, for certain animal species, there is no genetic adaptation to mutations from low-dose, chronic, artificial radiation exposure.[47]

4) EPA should not only be concerned about cancer experienced by the *initially* exposed generations, but also about *transgenerational cancer* that comes from this initial exposure. In addition, EPA must consider endpoints other than cancer, both in

the exposed generation and future generations. EPA should not assume that, although cancer may carry the greatest impact most immediately, genetic and epigenetic impacts won't surpass this risk in a few generations. We are already four generations into release of, and continual exposure to, artificial and "technologically enhanced" natural radioactivity with no end in sight. Our genome could be reaching a tipping point. EPA needs to take this opportunity to be creative, using what data are available, and integrate protection for the most vulnerable from negative non-cancer impacts. Yet ICRP doesn't model for genetic impact past the second generation. The RAC should help guide EPA to protect for impacts of radiation exposure well past what the ICRP model allows because we are dealing with radioactive materials that will remain dangerous for many human generations.

ICRP 103[48] discusses the multigenerational genetic impacts of radiation exposure but stops calculating risk past the second exposed generation. In the second generation exposed, ICRP calculates as many as 6700 people in one million may get a genetically induced disease from a dose of 1 Gray. This number is higher than the 4700 in a million for the first generation. But the ICRP numbers fail to account for damage past the second generation because "the risk estimates presented for the first two generations adequately reflect the current state of knowledge in this evolving area."[49] In other words, we don't know enough to predict further than two generations. Again, the ICRP model falls short of the reality we will be dealing with. The risk is there, but the model fails to tell us what it is.

There is mounting evidence that epigenetic/non-targeted effects, which were unrecognized as conveyers of radiation damage traditionally, can also cause disease. These are generally defined as effects that occur from radiation exposure and which are not attributable to direct genetic damage–that is, direct damage to the primary DNA sequence, [50] rather than damage or changes to or through methylation, chromatin, histone covalent modification and chromatin structure.

These effects can be manifested in unirradiated offspring as increased cancer predisposition, increased mutation rates, decreased fertility rates, and a radiation-induced increased sensitivity to further mutation from a variety of insults, not just radiation. These effects indicate a general destabilization of genomic integrity through several generations

subsequent to the one initially exposed.[51] Other effects may include cardiovascular, gastrointestinal and respiratory diseases.[52] In a world continually contaminated by man-made radioactivity, increasing rates of disease could beset an increasingly radiosensitive population. Experts say we can start assessing implications for human health, despite needing more human data. [53], [54] For more information on the topic of epigenetic impact of radioactivity and genetic modeling, see additional references in my comments submitted on behalf of Beyond Nuclear to EPA ORIA during the first round of comments on this ANPR.

5) To implement any protection standard, EPA must start exploring additional methods of assessing radiation damage since, historically, estimates of dose have been riddled with uncertainty. RAC should point to concrete ways EPA can combat the "magical thinking" that believes dose estimation is much more reliable than it actually is and to use new biomarker techniques to help deal with the uncertainties. The belief in dose estimates remains steadfast—even if radiation-associated diseases increase in a population, unreliable dose estimates are used to claim radiation is not responsible. These dose estimations have often been based on averaged radiation releases measured by industry. Measurement of these releases has been fraught with secrecy and improper measurement technique, resulting in unrealistic exposure scenarios. One concrete remedy for this uncertainty is to compel regulations on radiation exposure to come into line with other EPA risk levels and recommendations on toxins, such as complying with recommendations in the EPA 2005 Children's Health Guidance and EPA Supplemental Guidance for Early-Life Exposure to Carcinogens; and to make radionuclides subject to EPA's 10^{6} to 10^{4} risk factor as a group.

Assessing damage to individuals and populations from radioactivity often starts with calculating a dose to see if exposure was high enough to result in a disease. There are a number of problems with this technique.

First, we no longer exist in a world without man-made or technologically enhanced radioactivity. Therefore, in order to be fully protective, EPA must set standards that account for impact of previous artificial and natural radiation exposure whether or not EPA can regulate exposure to those sources. This includes past releases from nuclear facilities. NRC

claims the average member of the public receives 620 millirem per year of radiation. Voluntary or not, the risk in the NRC number is already there and must be added to whatever EPA sets as an allowable further exposure. While EPA ORIA doesn't have the authority in this context to set standards for all nuclear facilities or exposure pathways EPA must still account for current NRC exposure rates in its regulations.

Second, radioactive releases from nuclear facilities are averaged over a much longer time than their actual release duration. EPA must not allow annualization or dose averaging of batch releases from nuclear facilities. There is evidence that this averaging gives a false picture of childhood health damage from radiation because exposure during certain nuclear operations could be higher for a shorter time. "The normal emission concentration… is about 3 kBq/m^3, but during inspection/refuelling [sic] … this concentration abruptly increased to ~700 kBq/m^3 with a peak of 1,470 kBq/m^3. In the following days…the concentrations of released radioactive noble gases were still much higher (average = 100 kBq/m^3) than during normal power operation"[55] This batch releasing points to a mechanism whereby child health could be affected more quickly during very vulnerable developmental stages, causing a negative health impact that would not have been expected. Such a phenomenon could be used to explain the increases of childhood leukemia around nuclear facilities in France, Germany and elsewhere.

Third, industry measurements of radioactive effluent, often the only ones available, are notoriously incomplete and uncertain, especially for "unplanned" and batch releases.[56] NRC admits in its 2006 final task force report on radioactive releases that "The task force did identify that under the existing regulatory requirements the potential exists for unplanned and unmonitored releases of radioactive liquids to *migrate offsite* into the public domain *undetected*."(emphases added) and "Currently, the NRC lacks regulatory guidance for monitoring and evaluating both the immediate and long term offsite dose or environmental impact of these inadvertent releases."[57]

Tritium has been a large component of unplanned leaks across the nuclear fleet.[58] Recently, tritium has been found in ice at New Jersey's Hope Creek nuclear facility and also at nearby Salem at levels 500 times greater than allowed by already non-protective federal water quality standards. As of the discovery, no obvious release pathways were identified.[59] But

uncontrolled releases are not the only concern: consider the instance of carbon-14. Before 2010, NRC licensees did not have to measure or report carbon-14 effluents of any type. Since 2010, licensees have to report c-14 effluents ONLY if they are considered a "principle radionuclide" meaning that they constitute a certain percentage of the radioactive releases of a reactor or a certain amount of the human dose. Carbon-14 (half-life over 5,000 years) releases (in the form of CO-2 and methane) do not have to be measured, however, but may be estimated by the licensee.[60] As stated earlier, both tritium and carbon-14 can collect in developing fetal tissue at twice the concentration compared to the tissue of the pregnant female. Yet, historical and continuing releases of these two nuclides, their ability to move ubiquitously in the environment, long hazardous lives, and the potentially underrated biological damage from their internally deposited beta particle radiation, have been largely downplayed or ignored altogether. This must change.

ALL radionuclide releases should be regulated. EPA can, in part, remedy the lack of reliable data from industry by establishing and/or encouraging independent real-time monitoring of releases at stacks and pipes, biomonitoring, and more robust and appropriate environmental sampling efforts (see below). It is magical thinking to suggest that micromanagement of released artificial radioactivity exists. This is why all radiation leaving a nuclear facility should be accounted for at the point of release such as the stack or pipe, and the public and regulators should be notified ahead of time of refueling or batch releases. The public should know, in real time, what is being released. However, once radioactivity is released to our environment, it is silly to base any regulation on assumptions that we can exercise any control over it at all. In reality, EPA, NRC or any regulatory agency has little control over which radionuclide goes where, or by what pathway it may enter the human body. Not only is there little control, estimating the impact is also fraught with uncertainty, particularly across generations. In the current regulatory regime, the best protection EPA can offer an unwitting public is protection of the most vulnerable and to undertake public right to know with regard to all radioactive releases.

Biomarkers and environmental measurements

One way to curb magical thinking and to understand what is actually occurring in humans and their environment is to encourage widespread biomonitoring and other measuring

efforts. Already-released carbon 14, cesium-137, strontium-90 and any other long-lived radionuclide must be measured in the environment (with the proper caution, tree rings and other media can be examined for this) and be counted as a dose across generations (not just the T1 generation). This is now baseline and must be deducted from present and future allowable releases for as long as the nuclides remain active.

In 40 CFR 190, EPA briefly mentions that "measureable concentration or activity level[s] have been used in the past, in addition to dose and risk, as part of an exposure assessment. To this end, EPA should add to its regulations as proof of exposure, bio markers such as chromosome rings and dicentrics, protein markers and other biological impacts, and should work with public health groups and exposed communities to establish independent methods to monitor these impacts on a regular basis and in consort with batch releases. There is scientific precedent for such monitoring both around German facilities and after the Three Mile Island accident, although neither was a protracted project.§

It is vital that, if EPA decides to update its 1977 radiation exposure regulations, it has technical support from RAC that includes the latest information on early life stage impacts of exposure to radioactivity. Where information is contradictory or incomplete, EPA must err on the side of fully protecting vulnerable life stages—a position supported by a number of Federal frameworks and guides. Unless EPA's new standards are fully protective of early human life stages, updating these standards will be inadequate and early human life stages, particularly females, will continue to disproportionately bear the burden of radiation exposure.

[1] Children's Health. Early Life Stages. USEPA http://www2.epa.gov/children/early-life-stages (accessed 3/22/15)
[2] http://www2.epa.gov/children/early-life-stages
[3] Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. http://www2.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf
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§ For a more complete discussion of biomarkers with references, see Beyond Nuclear comments to NAS Phase I Committee to assess cancer risks around Nuclear Regulatory Commission licensed facilities.

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